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TISSUE MECHANICS AND ITS RELATIONSHIP TO ATHLETIC INJURY PREVENTION

A PAPER PRESENTED TO THE 1990 AAHPERD CONFERENCE, NEW ORLEANS KINESIOLOGY ACADEMY MARCH 30, 1990

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ABSTRACT

Three types of dense fibrous connective tissue will be described: articular cartilage, tendon and ligament, and the characteristics of each will be related to their function. Articular cartilage is the smooth covering over the surface of bone at joints, and is comprised of up to 80% water. This smooth covering serves to decrease the friction between the surfaces in synovial joints, as well it serves as a cushioning and shock absorbing surface in the joints of the lower limb. Overuse activities and the aging process will cause damage to articular cartilage, producing pain, loss of joint mobility and eventual damage to bone. Tendons and ligaments are similar in structure, being comprised primarily of collagen fibres arranged in parallel bundles. These collagen fibres are embedded in a fluid matrix, along with chonrocytes and proteoglycan molecules. Overuse activities and the aging process will cause inflammation and weakness of the collagen bundles, resulting in strain injuries or chronic inflammation. Strenuous training programs which include eccentric exercise and deceleration activities may strengthen ligaments and tendons and prevent injuries.

This paper will attempt to describe the structure and function of three types of dense fibrous connective tissue, and their relationship to injuries. These include articular cartilage, tendon and ligament, which are often injured in sports activities. It has been suggested that exercise programs which selectively strengthen these structures may assist in injury prevention (Curwin, & Stanish, 1984; Henning, 1986; Stanish, & Curwin, 1989).

Cartilage

Cartilage is a specialized connective tissue which is stronger than ligament and tendon as it is modified to bear weight, but the structure consisting of collagen fibres, matrix and proteoglycans is similar to other dense fibrous connective tissues.. The articular cartilage found covering joint surfaces is not as strong as bone, as it is less rigid and somewhat deformable.

There are three types of cartilage found in the body: hyaline (articular) cartilage, fibrocartilage, and elastic cartilage. Hyaline cartilage is also called articular cartilage, and is found covering the articular surfaces of bones at joints, in the nose, at the ends of ribs. . Fibrocartilage is a specialized type of cartilage which is found in some joints, and functions in shock absorption and for deepening the socket of joints. This includes the menisci of the knee joint and the glenoid labrum of the shoulder joint. Elastic cartilage is found in only two locations in the body: in the ear and in the larynx, and is highly specialized for its role in these two organs..

Hyaline (articular) cartilage

As with other skeletal connective tissues, articular cartilage consists of cells and matrix. The cells are called chrondrocytes (chrondro=cartilage), which secrete the matrix , or the gel-like fluid that they live in. The matrix, or ground substance consists of water, salts and proteoglycans, which are molecules consisting of protein (5%) and carbohydrate (sugars) (95%). (Mow, 1989) The most important characteristic of proteoglycans is the ability to bind water, to alter the stiffness of the matrix. Hydrated proteoglycans form the main components of the highly viscous and mucous-like fluid which comprises the matrix of the intercellular ground substance of all skeletal connective tissues. This matrix is a thick fluid and would be hard to pour, with the thickness being dependent on the property of the proteoglycan to bind water. The more water that is bound by the proteoglycan, the more firm the tissue becomes, so the tissue can become progressively more resistant to compression. The water increases the resistance to compression of tissues, so that a tissue that has absorbed more water will be firmer and provide stronger support(Mow, & Rosenwasser, 1987). The water is also important for diffusion of nutrients to cells through the intercellular matrix.

Connective tissues often receive their nutrition from direct diffusion from the intercellular fluid, as substances move from high to low concentration across the matrix. Diffusion of nutrients occurs down the concentration gradient, from the blood vessels through the intercellular fluid to the cells.

Connective Tissue Fibres

The connective tissue also contains a number of long protein fibres which are found in the matrix around the cells, and function to contribute to the strength of the tissues. There are three types of protein fibres: collagen fibres, reticular fibres, and elastic fibres. Collagen fibres are fibrous structural proteins found in the tissue matrix to provide stiffening. Collagen fibres are relatively stiff, and they can elongate to only 5% of their resting length before failure occurs (Butler, Grood, & Noyes, 1978). The collagen fibres consist of a regular structural arrangement of protein molecules which have been woven together in bands, so the fibres are strong and relatively inelastic. The more collagen fibres that a tissue contains, the more tensile strength it has, and the more resistant to strain or elongation. A tissue with a large collagen component is relatively inextensible, as it will not stretch appreciably when pulled. When an athlete increases the strength of connective tissues, this is usually due to an increase in the amount of collagen in the tissue.

Each collagen strand consists of a 4 to 1 stagger of tropocollagen molecules. Tropocollagen consists of a string of polypeptide chains, held together by electrostatic bonds (Butler, et al., 1978; Mow, 1989). The strings of collagen are joined together end to end in a splicing fashion; as well they are joined laterally by side to side bands. The strength of the collagen depends on both the end-to-end bonds, as well as the side-to-side bonds, giving the collagen strands the ability to resist tension, or elongation. The more collagen fibres that a tissue contains, the more tensile strength it has, and the more resistant to strain or elongation. A tissue with a large collagen component is relatively inextensible, or it doesn't stretch appreciably when pulled. When an athlete increases the strength of connective tissues, this is usually due to an increase in the amount of collagen in the tissue, which will also increase the number of side to side bonds (cross-links), as well as end to end bonds(Curwin, et al., 1984). Activity will also change the architecture of the arrangement of collagen fibres in response to the direction of the applied stresses. For example, if a ligament or tendon is constantly subjected to tensile forces, the collagen fibres will line up in parallel bundles in the same direction as the applied force. This arrangement will provide greater resistance to the applied forces, as both the amount, and the direction of the fibres has adapted to the applied stress.

The collagen fibres in tendon will also atrophy with underuse, as if a subject is casted for 6 months, the fibres will decrease in size. If the tension is removed from tendon, the collagen fibres will disorganize and the fiber directions will be random. Healing by use of a cast, or by use of massage or ultrasound in which no tension is placed on the fibres will not produce strong, well organized parallel fibres (Stanish, et al., 1989).

The matrix is quite firm, yet resiliant, as a large portion of the matrix is comprised of proteoglycan which is bound to water to form a gel. The more proteoglycan in the tissue, the less compressible as the water content of the tissue increases and fluids are incompressible. Proteoglycans are formed primarily from carbohydrate molecules, which have the ability to bind a large amount of water (Buckwalter, Hunziker, Rosenburg, & Coutts, 1987a).

Hyaline cartilage contains a large proportion of water, so it is relatively incompressible and difficult to deform. This is desirable to absorb forces and withstand impact shock. The high water content of articular cartilage is also important to diffuse nutrients to the tissue, since cartilage is avascular and nourishment does not come directly from the capillaries. The water contained in hyaline cartilage is important in the diffusion mechanism; however with age the diffusion rate decreases because the percentage content of water also decreases This presents a problem when cartilage injury occurs in older adults, as healing occurs very slowly, or not at all. Cartilage cells have a slow metabolic rate, so that healing and growth of these tisues always occurs at a slow rate. Collagen fibres are also present to add strength to the articular cartilage.

Hyaline cartilage is found at the ends of bones, to reduce the friction in synovial joints. There is no perichondrium, or layer of fibrous connective tissue protecting the articular cartilage of joints, so the cartilage is naked at the joint surfaces. This produces an efficient bearing surface in which the coefficient of friction is less than .002 between the cartilage surfaces with the synovial fluid.

Synovial fluid is found in the joint capsules of synovial joints, and has a composition similar to blood plasma. It assists in the nutrition of the articular cartilage, as nutrients from the blood are carried via this fluid into the articular cartilage (Fulkerson, Edwards, & Chrisman, 1987).

Articular cartilage may be from 1-7 mm thick, depending on the joint, and consists of three layers. The superficial layer is one in which the chondrocytes are flattened to produce a smooth surface of low friction. The collagen fibres are also arranged in layers parallel to the surface of the joint, and the layers are thicker and denser in the superficial region. The cells in this zone may be an important area of proliferation, in which some of the long bone growth actually occurs, especially in growing children. The middle layer of the cartilage consists of larger chondrocytes, indicating hypertrophy of these cells. The cells become longer and are structured in columns which

run perpendicular to the surface. The collagen fibers in the middle layer are also structured in a more random arrangement, as the fibres change their orientation from horizontal to vertical as they approach the deep layer. The fibres are also less dense in this layer, as much of the proteoglycan and the associated water are stored in this layer (Buckwalter, et al., 1987a; Fulkerson, et al., 1987; Mow, et al., 1987; Mow, 1989)

The deep zone is the zone of calcification, in which the cartilage cells begin to calcify in several layers of increasing hardness. The vertical cartilage cells interlock with the bone cells right below at a darkened area known as the tideline, which is the zone of ossification of the cartilage. The vertical cartilage cells first become fibrocartilage, then mineralized fibrocartilage, and finally become part of the cortical bone at the surface of the bone-cartilage interface at the tideline. The transition of cartilage to bone occurs gradually, so the absorption of forces at joints can occur over a greater distance before bone is encountered. This is a very mechanically sound structure, as the forces of impact can be dissipated over several layers of increasing stiffness before the actual bone is reached (Fulkerson, et al., 1987).

Biomechanics of articular cartilage

Articular cartilage is a viscoelastic material, which means that it is both elastic, so that it can regain its original shape after deformation; as well as being viscous, in that the deformation is related to the strain rate.

Stress and Strain

Stress is the force developed in the tissue when you try to deform it, and is the amount of force per unit area of tissue. Stress is equal to the applied force divided by the cross sectional area., and is measured in Pascals, or Newtons per square meter (N/m2); or Megapascals, Newtons per square centimeter (N/cm2) (Butler, et al., 1978; Nordin, & Frankel, 1989). Stress is normalized to the size of the specific tissue that is being tested. Mechanical strain is the actual deformation that the tissue undergoes when forces are applied to it. The physical deformation of the tissue due to these applied outside loads can be measured as a percentage of resting length, or percentage strain.

The stress-strain curve is the mechanical representation of the relationship between stress and strain for any given tissue. As the stress is increased, the amount of deformation occurring in the tissue will also increase. The linear region of the curve occurs when stress and strain are directly proportional, and usually occur within the biological range of the tissue. This is known as the elastic region of the curve, as following these deformations the tissue will normally return to resting length. The elastic limit is the portion of the curve in which the response is no longer linear, and the tissue begins to deform plastically so it will no longer resume the uninjured state (Butler, et al., 1978; Nordin, et al., 1989).

The Viscoelastic Response

The response of connective tissue to loading is not perfectly linear or elastic, but it is a viscoelastic response. There are different forces applied to a ligament depending on how fast it is loaded. Viscoelasticity is often defined as the property of a material to show sensitivity to rate of loading or deformation. Creep and relaxation are two characterisitics of viscoelastic materials. During creep tests, the load is suddenly applied and kept constant thereafter, and the continuous displacement is recorded against time. In relaxation tests, a deformation is produced and then fixed, and the resulting decrease in load on the tissue is recorded as a function of time (Butler, et al., 1978; Nordin, et al., 1989).

The elastic response of the tissue is often represented as a spring; as when the stress is increased there will be a linear deformation. Young's modulus, e, is the ratio between stress and strain, or it is the slope of the curve and is a measure of the stiffness of the tissue. Another element which can be used to model the behaviour of tissues is the dashpot, which is a viscous element. Stress is linearly related to strain rate, or how fast the tissue is being deformed. The velocity of the deformation of the tissue in compression or tension will determine the viscosity coefficient, or the slope of the curve. This is also related to how thick the fluid is that you are moving through. A biological tissue will often withstand greater stresses at higher strain rates, so that a greater load can be applied before failure at high strain rates.

Ligaments have a mechanical response similar to that seen in other nonlinear viscoelastic soft tissues. The load-deformation curve for ligaments consists of a linear toe region, due to the straightening out of the crimp in the collagen fibres. This is followed by a nonlinear elastic region, in which the ligament responds to increasing loads by deforming to an optimal length of 7-8% of resting length before failure occurs (Butler, et al., 1978).

The area under the stress-strain curve is the energy stored by the tissue during the deforming episode. If the tissue is deformed by a certain load, and then allowed to return to resting length, the area under the curve would be the energy stored by the tissue. If some of the energy is lost as heat, this would show up in the decreased slope of the return curve. The difference between the deforming curve and the return curve is known as hysteresis, and represents the amount of energy lost as heat from deformation to reformation of a loaded tissue.

Cartilage in the Aging Process

It has been suggested that the articular cartilage of children is softer and less stiff than that of the adult (Micheli, 1986) This is due to the larger

amount of immature collagen in the cartilage of children, which will decrease the strength and stiffness of the tissue. As well, the immature proteoglycan molecules are less able to take up water to increase the stiffness of the tissue, also decreasing the strength and stiffness. It has been suggested that prepubertal athletes should not run long distances, as the softer articular cartilage may lead to early degeneration of the tissue (Micheli, 1983).

. Cartilage is subject to changes with the aging process, as the cells stop multiplying in the adult and you have a constant number of cells. The chrondrocytes no longer continue to produce the tissue components, and the tissue becomes inert. As cartilage wears out, there are fewer cells, but more matrix, so the matrix becomes thicker. Nutrition to cartilage is difficult at best, but it decreases with age due to the increased thickness and the greater difficulty encountered with diffusion. The deepest part of the cartilage will die, producing a layer of dead cells, which will decrease the water content of cartilage and decrease the resistance to compression. The cartilage becomes more brittle, and no longer resists compression in the same way. There is a decrease in the amount of proteoglycan with aging, so the amount of water in the cartilage decreases.

This decrease in water content decreases the firmness of the cartilage, so it becomes softer and less turgid. There is greater calcification of the cartilage, as the fluid content of the tissue decreases, the calcium found in all body fluids is left in the cartilage, so it tends to become more brittle with a greater probability of damage during exercise (Buckwalter, et al., 1987a; Mow, et al., 1987; Mow, 1989).

Osteoarthritis

Osteoarthritis is a disease of hyaline cartilage, caused by excessive wear and tear on the joints with aging or overuse. The articular cartilage damage seen in young athletes such as Little league pitchers is a type of overuse injury, in which the softer cartilage of the young are unable to withstand the repeated trauma of intensive pitching.

In the older person, osteoarthritis is a degenerative joint disease caused by alterations in the structure of the tissue due to the aging process. As the amount of fluid in the tissue decreases due to the decreased amount of proteoglycan, the matrix decreases in volume, and this leaves the collagen fibres exposed at the joint ends. This is known as fibrillation of the cartilage, and increases the friction at the joint considerably. Bone spurs may also be formed at the edges of the fibrocartilage layer of the articular cartilage, due to the increased friction in the joint. These injuries to the joint surface are poorly repaired, due to the poor blood supply and the resulting poor supply of nutrients and oxygen to articular cartilage (Buckwalter, et al., 1987a; Fulkerson, et al., 1987).

Dense Connective Tissue-Tendons and Ligaments

Tendons

The connective tissue found in tendons and ligaments is known as dense, fibrous connective and has a specific arrangement in which the collagen bundles are arranged in parallel. A cross-section through a tendon will illustrate the bundles of collagen fibres. Tendons are fibres which contain a large amount of collagen, in which all the bundles are in perfect parallel array. Tendon is the tissue which contains the largest amount of collagen of any tissue in the body (Curwin, et al., 1984; Stanish, et al., 1989). The bundles of collagen fibrils, or fascicles, consist of fibrils of different sizes. The larger the diameter of the fibers, the more collagen and the stronger the tendon will be. If there are a large number of smaller fibers, this will imply that the tissue will be weaker. Also, the more crosslinks between the collagen fibers, the greater the strength of the tendon. Due to the large amount of collagen in tendons, they are inextensible so the muscle forces are transmitted directly to the bone. All the fibres are oriented along the long axis of the tendon, to maximize tensile strength. The tendon primarily resists force in one direction, which is along the long axis of the tendon in the direction of the muscle shortening and lengthening. Thus the tendon is able to accomodate only to unidirectional forces, and has little resistance to forces in other directions.

Collagen fibres, hence tendons, are relatively inextensible so that when the muscle contracts the tendon will resist this pull and the force is applied to the bony insertion. If the tendon was too elastic, when the muscle contracted the force of the muscle would be absorbed in tendon deformation. Muscles are relatively elastic compared to tendons, but this is mostly due to the connective tissue surrounding the muscle cells. The structure of collagen is designed to resist stretch, so tendons are relatively inelastic.

The structure of tendon includes a large amount of collagen, the ground substance, plus some cells including fibroblasts or fibrocytes which are the fibre-producing cells. The ground substance of the tendon is the gel-like matrix that fills in the spaces between the collagen molecules. The fibroblasts actually secrete the cells and the amino acid molecules which make up the collagen fibres of the tendon, so the activity of the fibroblasts will actually increase the size of the tendon. The ground substance of the tendon also contains a large number of proteoglycans, consisting primarily of glycosaminoglycans (GAGS). The exact role of the GAGS in tendon remains unclear, although they appear to give the tendon collagen molecules an adhesion quality.

The tendon appears white in colour, due to the large number of white collagen fibres. The collagen molecules are linked together, as there are three amino acid strands in each molecule. These three linked molecules can also be linked to neighboring molecules by a system of cross-linking, which is

very important in tendon strength (Curwin, et al., 1984; Curwin, Vailas, & Wood, 1988). If animals are fed a diet which prevents the production of crosslinking, there are significant losses in tendon strength. There are some capillaries associated with the tendon, which usually run along the outside edges of the tendon as the middle of the tendon has a poorer blood supply than the periphery. However, these tissues have a generally good blood supply, and this vascularization is accompanied by ample nutrition which implies better healing of injuries. This blood supply generally ensures that healing of the tissue will occur with time. There is also a nerve supply to the tendon, with the nerve trunks being located in the sheath of the tendons, and some of the nerve branches are found in the ground substance between the collagen fibrils.

It is important structurally to have the tendon firmly attached to bone, so there is a strong connection between these two structures. The tendon-bone connection is a mixture of the properties of bone and tendon, with a gradual transition through several zones from tendon to more cartilaginous and bony substances (Buckwalter, Maynard, & Vailas, 1987b; Woo, Maynard, Butler, & Lyon, 1987). As bones increase in size during growth, and the bones gradually ossify, the tendon becomes embedded in bone to form a strong connection. The collagen fibres of the tendon actually protrude into the bone, then the bone calcifies around the tendon fibres.

Stress-Strain Characteristics of Tendon

The stress-strain curve for a typical tendon has been reported by several authors (Butler, et al., 1978; Curwin, et al., 1984; Nordin, et al., 1989). The curve consists of a linear toe region, in which very slight stresses (loads) will produce large elongations. This point on the curve is due to the wavy pattern of the collagen fibers, also known as the crimp. The initial deformation occurs as these fibres straighten out under the applied load and lose their wavy pattern. This is followed by a non-linear region, in which there are microtears occurring in the collagen fibrils but the structure appears to be intact. This is the case which occurs in tendinitis, in which there are microtears occurring in the collagen fibers, but complete failure has not occurred. Ultimate failure occurs at the point of maximum stress to the tissue, and following this point failure of the tendon occurs as the successive bundles fail.

In the case of tendinitis, the microtears have occurred in the tendon fibers, and often pain has resulted. The athlete will often decrease activity levels, and the tendon will begin to heal. Healing occurs when new collagen is formed to replace that which has been injured. In the normal regeneration of tendon, old collagen is being absorbed, and new collagen is being formed to replace it. It is possible to influence, through the rehabilitative process, how that new collagen is laid down. If the healing process is accompanied by the

application of mechanical forces to the tendon, the collagen will organize to best resist these forces.

The muscle itself also contains a large amount of collagen, in the muscle sheaths surrounding the bundles of muscle fibres and the myofibrils. This collagen also will organize to withstand the forces applied.

The Musculo-tendinous junction

The musculo tendinous junction is the junction between the tendon and the muscle to which it is attached. This has been found to be an area of relative weakness in the muscle-tendon unit, as this is the site of the majority of muscle strains. This junction consists primarily of collagenous connective tissue invested with fingers of muscle tissue (Garrett, & Tidball, 1987).

Tendon-Bone junction

The junction of the tendon to the bone consists of several layers, of increasing stiffness, as the tendon tissue becomes more like bone in structure. The collagen fibrils gradually become fibrocartilage, mineralized fibrocartilage, and then bone. At this junction between the tendon and bone, there is a change in mechanical properties from the more elastic tendon to the stiffer mineralized fibrocartilage. This may produce areas of stress concentration, or int asymmetries in the application of forces from the tendon to the bone. These areas of stress concentration are almost always the sites of injury to the muscle-bone system. It has now been suggested that up to 100% of muscle injuries occur at the muscle-tendon junction (Tidball, & Chan, 1989)

Mechanisms of Injury to Tendon

The most common injury diagnosed in tendon is tendinitis, or a chronic inflammation of the tendon. There are many unanswered questions regarding the biomechanics, or pathomechanics of tendon injury--why and how do these injuries occur? The causes of tendinitis are largely mechanical, although some other metabolic factors such as diet may also assist (Curwin, et al., 1984; Stanish, et al., 1989). There are two types of mechanisms which may produce tendinitis: internal (from within) and external (outside) forces. Internal forces, or tension related forces, result when the muscle attached to the tendon produces very large contractile forces , producing tensile forces which are which are transmitted to the tendon--the chronic overuse syndrome. External forces are those situations in which the tendon is contacted by the surrounding structures such as ligaments, bony apophyses, or tendon sheaths, and the stresses are due to these external forces.

A type II tendinitis, produced by muscle forces, is thought to be due to high forces of muscle contraction, often with the addition of other external forces as in landing or hitting a tennis ball. This often occurs when a muscle which is already contracting is suddenly elongated, producing sudden-onset tendinitis. The mechanism which appears to produce tendinitis is sudden

eccentric contraction of the muscle to which the tendon is attached (Curwin, et al., 1984; Stanish, et al., 1989) Eccentric contractions most often occur during deceleration of the body; as in landing from a jump, changing direction rapidly, or in running backwards. In backwards running, for example, the gastrocnemius/soleus muscle group is lengthening to decelerate the body with each step. These eccentric contractions produce very large forces on the Achilles tendon, which often produce tendinitis. A common situation for Achilles tendinitis is a basketball player landing from a jump and experiencing a sudden pain in the Achilles tendon, especially after a break in training.

Another common mechanism which produces tennis elbow, or tendinitis of the common extensor tendon occurs when the tennis ball strikes the tennis racquet in a weak backhand stroke. The elbow is often bent, and at impact the ball on the strings drives the wrist into flexion, putting an extreme stretch on the strongly contracted extensors. This eccentric contraction force is added to the concentric contraction already occurring, and the tendon forces approach the limit (Curwin, et al., 1984; Stanish, et al., 1989). The maximum stress for mammalian tendon is thought to be about 100 MPa (megapascals). The magnitude of these forces on the tendon increases with the activity: walking, jogging, sprinting, jumping. The forces produced in sprinting are extremely large, and the forces in the Achilles tendon reach a maximum at toeoff, which is near the point of maximum elongation. (Curwin, et al., 1984; Stanish, et al., 1989) has reported the tension in the Achilles tendon during sprinting approaches ten times body weight, with other activities such as jumping somewhat lower.. The skill of kicking also produces very large forces on the patellar tendon during rapid knee extension.

Eccentric contractions are produced in many of the phases in the performance of a sport skill It has been reported that the forces produced by these eccentric contractions approach the maximum tensile forces on the tendon. It has been suggested that it is not possible to maximally stress the muscle-tendon unit during concentric contractions; but that this occurs only during eccentric contractions. It has been recently stated that it is impossible to rupture a muscle-tendon unit using only concentric forces (Curwin, et al., 1984; Stanish, et al., 1989). In the skill of baseball pitching, for example, eccentric contractions occur just prior to the end of the backswing, to decelerate the backward motion of the limb prior to the forward motion of the throw. Eccentric contractions occur again following release of the ball, to decelerate the throwing arm gradually to prevent injury to the joint. The shoulder lateral rotators, the forearm supinators and the back extensor muscles are all contracting eccentrically to decelerate the body parts following the throw.

Type I tendinitis occurs when there is impingement of other body structures or external objects on the tendon. An example of this is

supraspinatus tendinitis, in which during abduction of the shoulder joint the tendon impinges on the coracoacromial ligament, producing tears in the tendon. This type of injury is commonly seen in swimmers, as well as in throwing athletes. Swimmers often also have chronic shoulder instability along with the tendinitis, so it is difficult to determine the actual causes of pain. The coracoacromial ligament is very sharp on the lower edge, making the pressure on the tendon even larger. Healing of this tendon is also impeded by the very poor circulation to the area , and if the blood supply is poor the metabolic rate also has to be poor. This produces very poor and very slow healing to this area, which is often very frustrating to patients. Also, the tendon is compressed during adduction, it is wrung out by the compression onto the ligament, reducing the blood flow to the area even more. These impingement syndromes are often seen in throwing athletes, as have been described by (Jobe, & Nuber, 1986). However, rotator cuff tears can also be produced by eccentric contractions in throwing athletes, so can also be produced in tension-related situations.

Treatment of these chronic shoulder tendinitis problems consist of strengthening the tendon using eccentric contractions in an exercise program of increasing intensity. The use of slow eccentric contractions is followed by faster and more forceful eccentric contractions, such as those produced by catching a small weight dropped from a height.

These chronic impingement syndromes are best treated with rest and avoidance of the activity which causes the pressure; followed by a re-training program when the inflammation has subsided. If the inflammation persists often surgery is required to remove the impingement. In the case of the rotator cuff, this is often an acromioplasty, or thinning down of the acromion process. This should be accompanied by release of the coracoacromial ligament, which also impinges on the tendon superiorly. The chronic rotator cuff tear to the shoulder can be diagnosed by moving the shoulder joint into ninety degrees of flexion, accompanied by internal rotation, which will produce a painful arc. This position moves the supraspinatus tendon directly under the acromion.

Type I tendinitis of the Achilles tendon is also produced by certain types of shoes with a strap on the back, which will place high pressure on the tendon, and cause inflammation. If this pressure is continued over time, scar tissue will form in the tendon, and chronic stenosing tenosynovitis will result. Another mechanism of producing tendinitis is as an overuse injury, in which repeated overuse of the tendon will produce microtears, which will eventually produce chronic pain and inflammation. This will also produce a sensation of tightness in the muscle-tendon unit, which often accompanies tendinitis.

Fatigue and Tendinitis

Another possible cause of tendinitis may be fatigue in the muscle or tendon unit. It has been suggested that tendons may be injured during the dehydrated state following intense exercise. If you decrease the water content of the tendon, it will become stiffer and more able to bear loads. However, the disruption of the electrolyte balance within a muscle/tendon unit following fatigue of the muscle may promote injury to the collagen of the tendon. The mechanism for this occurrence remains unclear.

Stretching the Tendon

Although passive stretching has been suggested to decrease the incidence of tendinitis, there is little empirical evidence to back this up. A stretched position of the muscle may stretch the muscle, and the sheath of the muscle and tendon, but may not actually stretch the tendon. In order to create a stretch on the tendon, it must be done with the muscle active, as in eccentric contractions. This combines the maximum length with the maximum load, giving the highest stress to the tendon.

Strengthening the Tendon

In order to prevent these high stresses producing microtears and inflammation on the tendon, the tendon must be trained to withstand these high forces. The tendon must be strong enough and flexible enough to withstand the tension produced by high speed movements such as sprinting and kicking. This is done by overloading the tendon with high tensile forces such as seen during vigorous exercise programs. When the muscle is contracted, the tendon is under tensile loading in resisting this contraction, and transmitting the force of contraction to the bone. Strengthening of the tendon of an athlete must be done using high speed eccentric contractions such as those seen in sports activities (Stanton, & Purham, 1989). This suggests that re-training of these structures requires a much more strenuous program than has been used in the past., such as a program known as Eccentric Exercise Programs (EEP) (Curwin, et al., 1984; Stanish, et al., 1989).

The EEP should be used only in situations in which the tendinitis has resulted from tension situations. The severity of the symptoms must also be determined, to determine how much force should be placed on the tendon. Stanish and Curwin (Curwin, et al., 1984; Stanish, et al., 1989) have described a progressive strengthening program using eccentric contractions (EEP). This program progressively overloads the tendon to the point where the tendon is subjected to forces similar to those seen in sports activities. The athlete progresses from controlled lowering against the resistance offered by the therapist, to 'catching' a weight, or the weight of the body during rapid lowering. This rapid lowering increases in speed, and forces applied, as the program progresses. However, it was estimated(Curwin, et al., 1984; Stanish, et al., 1989) the forces on the Achilles tendon in this program to be only approximately 60% of those seen in high speed sports activities such as

sprinting. For example, in the treatment of Achilles tendinitis, the patient will stand on the edge of a raised stair and allow their weight to drop downward, then stop the downward drop with a strong contraction of the calf muscles, stressing the Achilles tendon. They referred to this as the 'drop and stop' phenomenon. They reported an improvement rate of over 90% in treating patients with chronic tendinitis; with over 44% attaining complete relief from their symptoms. The success rate varied with the type of tendinitis being treated, with tennis elbow being the most difficult to treat. Another 44% attained some relief from their pain; while 10% did not have any change in their symptoms. The final 2% were referred to an orthopedic surgeon for possible surgical intervention. The majority of the patients noticed some improvement in less than 5 weeks.

A similar program was devised by Stanton and Purham (Stanton, et al., 1989), in which eccentric exercises were used to strengthen the hamstring muscles following injury. The program was devised especially for sprinters, and consisted of quick active knee extension movements from a lying position with the lower leg free. The knee extension was rapidly decelerated via strong eccentric hamstring contraction, which was quickly followed by a concentric contraction. As the athlete progresses through the program, an additional weight can be added to the lower leg to increase the force of the eccentric contraction required.

The tension applied to the tendon should be applied in line with the long axis of the tendon, as if the forces are applied obliquely there will be assymetrical force application to the Achilles tendon. Tension on the tendon causes the collagen fibres to organize in parallel; if the tension is removed the fibres will become disorganized. If the tendon is subjected to compression, as seen in the supraspinatus tendon in swimmer's shoulder, the composition of the tendon will change and become more like a compressive tissue with the collagen fibres arranged in non-parallel fashion.

When a tendon has undergone strengthening, there is a greater proportion of collagen, which can be monitored by measuring the radioactive proline, a component of collagen. There is an increase in the size of the tendon, and increase in the amount of collagen, and most importantly, an increase in the chemical cross linking between molecules of the collagen. It was reported (Curwin, et al., 1988) that in growing animals, the tendon responded faster than the muscle to the overload situation by hypertrophy. Tendon also has a slower metabolic rate than bone in the adult subject, so does not regenerate as quickly. Healing is slower in the older adult, so more time must be taken for healing to occur.

Some experimentation is also being done in use of electrical stimulation to strengthen the tendon. Electrical currents are applied directly to the tendon, in order to enhance the tensile strength.

Tendon Injuries in Young Athletes

Many of the overuse injuries which occur to young athletes are apophyseal injuries, which are actually injuries to the growth cartilage. An apophysis is a prominent site where a tendon unites with a bone, eg. the greater tuberosity of the humerus, the tibial tubercle, and the epicondyles of the elbow. In the growing child, cartilage occurs between the tendon and bone, so the forces applied to the bone by the muscle-tendon unit are transmitted to the bone by an area of cartilage (Clain, & Hershman, 1989). This tissue is much less resistant to tensile forces than is bone or tendon, so there are avulsion lesions in the small areas of the developing ossification center in the anterior part of the tibial tuberosity (Ogden, & Southwick, 1976; Ogden, Tross, & Murphy, 1980).

The forces on the growth plates and the muscle-tendon units increase significantly during the adolescent growth spurt. (Micheli, 1986) has proposed that this rapid skeletal growth during puberty leads to relative 'tightness' of the soft tissues, as skeletal growth is faster than the elongation of the muscle-tendon units. This increased growth creates higher tensile forces on the muscles, tendons, and their attachment sites (the apophyses)

Two common apophyseal injuries of adolescence result from this increased tightness of the muscle-tendon units. Osgood-Schlatter's disease and Sever's disease (Clain, et al., 1989; Micheli, 1986). Since the growth plates around the knee will produce 65% of the growth of the lower limb, the insertion of the quadriceps tendon just below the knee is a site of increased tensile forces, producing Osgood-Schlatter's disease. In Sever's disease, there may be a 'tight' gastrocnemius-soleus complex as skeletal growth in the leg exceeds the rate of soft-tissue elongation. This may produce inflammation of the apophysis of the calf muscles (Micheli, 1986).

LIGAMENTS

Ligaments are the bone-to-bone connectors, also comprised of dense connective tissues, and are similar in structure to tendons. Ligaments consist of a close packed arrangement of collagen and elastic fibres running in two directions, much like a web. They act in a passive role in joint stabilization, and in guiding joint motion (Frank, Woo, Andriacchi, & Brand, 1987)

. Ligaments are structured to prevent unwanted motion in several directions, so a typical ligament will consist of parallel collagen fibres running in the direction of most of the forces, with elastic fibres at right angles to these fibres to produce lateral stability. The elastic fibres are yellow in colour, in contrast to the white collagen fibres, so the ligament will appear yellowish when compared to a tendon. Since the collagen fibres are relatively inelastic, they will restrict the undesirable movements at a joint such as seen in the knee and ankle. The lateral ligaments in these joints will restrict the sideways motion of the bones to maintain joint stability, but the elastic fibres

will allow the desirable motions such as the anterior-posterior motions at the knee and ankle (Buckwalter, et al., 1987b; Butler, et al., 1978; Carlstedt, & Nordin, 1989)

Ligaments have a mechanical response similar to that seen in other nonlinear viscoelastic soft tissues. The load deformation curve for ligaments consists of a linear toe region, due to the straightening out of the crimp in the collagen fibres. This is followed by a nonlinear elastic region, in which the ligament responds to increasing applied loads by deforming to an optimal length of 7-8% of resting length.

The area under the stress-strain curve is the energy stored in the tissue during the deforming episode. If the tissue is deformed by a certain load, and then allowed to return to resting length, the area under the curve would be the energy stored by the tissue. If some of the energy is lost as heat, this would show up in the return curve. The difference between the deforming curve and the return curve is known as hysteresis.

A few ligaments are different in structure in that they are largely elastic, with very little resistance to stretch. One example is the ligamentum flavum, which connects adjacent lamina and resists flexion gently like an elastic band. As forward trunk flexion is performed, the ligamentum flavum slows the motion down gradually so the end point of flexion is reached gradually. This action allows for gradual braking of forward trunk motion, which is safer for the vertebral column. Once the trunk is in a flexed position, the fibres are stretched and their elastic recoil will assist the return to the extended position. The ligamentum flavum is a noticeably yellow ligament, due to the high content of elastic fibres.

Ligament-Bone Junction

The attachment of the ligament to the bone occurs at the ligament-bone junction, through a series of zones. From the tendon fibers, the fibers enter a zone of fibrocartilage, mineralized fibrocartilage, and finally to cortical bone(Woo, et al., 1987). This provides a gradation of stiffness from the ligament to the bone, allowing for gradual transfer of energy at the junction.

Repair of damaged ligaments and tendons will occur in time, as they normally have an adequate blood supply. The replacement tissue for the damaged ligament or tendon is secreted by the action of fibroblasts which produce fibrous connective tissue. Tendons will usually regenerate with almost the same amount of elasticity as the original tissue, although the scarred area containing the replacement tissue may be more susceptible to recurring injury due to the slightly greater resistance to tension. A torn muscle is even more susceptible to recurring injury, as the scar tissue in repair is formed primarily from collagen which is less elastic than than the original muscle cells. This is also true in replacement of cardiac muscle which has been injured during a heart attack, as the injured cardiac muscle is

replaced by inelastic collagen scar tissue. The heart is then less elastic during both the filling and the pumping process, limiting the cardiac output of the patient.

If the tendon or ligament is completely torn, the ends will grow back together if they are close together following the injury, else the orthopaedic surgeon will place the torn ends close together during an operation. However, if the blood supply has been reduced in the injury the ligament may never completely heal, as often occurs in cruciate ligament tears. When a replacement ligament is required for a torn cruciate ligament, it is usually replaced by connective tissue from another site. The strength of connective tissues is difficult to replicate with artificial ligaments, so the replacement is usually one of the tissues of the injured athlete. A common replacement for anterior cruciate ligaments is a portion of the patellar tendon, as it may be the strongest connective tissue in the body .

Prevention of Injury to Ligaments

As with other tissues, injury prevention is related to increased strength, flexibility, conditioning and warmup and correct technique. One of the most common and serious ligament injuries is the tearing of the anterior cruciate ligament (ACL), which is a common sports injury. A program has recently been devised which may assist in prevention of injuries to the ACL (Henning, 1986). The ACL injury prevention program consists of three phases The athlete first learns the basic skills of accelerating around turns, two-step stops, and bent knee landings. These skills will decrease the forces on the knee joint during performance of these sport skills, and possibly improve the technique of the athletes in performing these skills. The athlete will then practice incorporating these skills in specific play situations which may produce ligament injuries. Finally, the athlete must be able to use these skills in a game situation. The author reported that the incidence of ACL injuries in three sports teams decreased by 2.5 times less after initiation of the injury prevention program.(Henning, 1986). Further testing and validation of this program is required to determine if this is a valid program for ligament injury prevention.

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